Germ cell migration in zebrafish is cyclopamine-sensitive but Smoothened-independent.

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Public Summary:

Scientific Abstract:
Primordial germ cells (PGCs) are the progenitors of reproductive cells in metazoans and are an important model for the study of cell migration in vivo. Previous reports have suggested that Hedgehog (Hh) protein acts as a chemoattractant for PGC migration in the Drosophila embryo and that downstream signaling proteins such as Patched (Ptc) and Smoothened (Smo) are required for PGC localization to somatic gonadal precursors. Here we interrogate whether Hh signaling is required for PGC migration in vertebrates, using the zebrafish as a model system. We find that cyclopamine, an inhibitor of Hh signaling, causes strong defects in the migration of PGCs in the zebrafish embryo. However, these defects are not due to inhibition of Smoothened (Smo) by cyclopamine; rather, we find that neither maternal nor zygotic Smo is required for PGC migration in the zebrafish embryo. Cyclopamine instead acts independently of Smo to decrease the motility of zebrafish PGCs, in part by dysregulating cell adhesion and uncoupling cell polarization and translocation. These results demonstrate that Hh signaling is not required for zebrafish PGC migration, and underscore the importance of regulated cell-cell adhesion for cell migration in vivo.